

Enhancing Hit Identification in *Mycobacterium tuberculosis* Drug Discovery Using Dual-Event Bayesian Models

Sean Ekins^{1, 2*}, Robert C. Reynolds^{3,4}, Scott G. Franzblau⁵, Baojie Wan⁵, Joel S. Freundlich^{6,7} and Barry A. Bunin¹

¹Collaborative Drug Discovery, 1633 Bayshore Highway, Suite 342, Burlingame, CA 94010, USA.

²Collaborations in Chemistry, 5616 Hilltop Needmore Road, Fuquay-Varina, NC 27526, USA.

³Southern Research Institute, 2000 Ninth Avenue South, Birmingham, AL 35205, USA.

⁴Current address: University of Alabama at Birmingham, College of Arts and Sciences, Department of Chemistry, 1530 3rd Avenue South, Birmingham, Alabama 35294-1240, USA.

⁵ Institute for Tuberculosis Research, University of Illinois at Chicago, Chicago, IL 60607, USA.

⁶Department of Medicine, Center for Emerging and Reemerging Pathogens, UMDNJ – New Jersey Medical School, 185 South Orange Avenue Newark, NJ 07103, USA.

⁷Department of Pharmacology & Physiology, UMDNJ – New Jersey Medical School, 185 South Orange Avenue Newark, NJ 07103, USA.

*To whom correspondence should be addressed. (e-mail: ekinssean@yahoo.com)

Running Head: Dual Event Bayesian Models

Figure S9. Results for the 194 compounds tested in the Selleckchem kinase library screened for whole-cell TB activity with Bayesian models. Random rate is based on the empirical HTS hit rate; MLSMR is based on the MLSMR dose response and cytotoxicity model; CB2 is based on the CB2 dose response and cytotoxicity model. Kinase is based on the MLSMR dose response and cytotoxicity model. Best curve is based on a 100% hit rate.

